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Control of Turf Brown Patch

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Credit to Miss Barbara Champlin for carrying out laboratory experimentations.

Cover illustration shows an active brown patch smoke ring and is courtesy of Mallinckrodt Chemical Works.

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A GLANCE AT THE BROWN PATCH PICTURE

Brown patch, or summer blight, is a major summer foliage disease on turf throughout much of the United States where high temperatures and humidities prevail for extended periods (8, 11). Brown patch is perhaps most damaging on fine turf in the area from New York and Philadelphia westward to St. Louis (12). Bent grasses, especially Colonial bents, are more seriously injured than the coarser fescues, blue grasses, wheat and rye-grasses (11, 14).

Brown patch is caused by a fungus, *Rhizoctonia solani* Kuehn (*Pellicularia filamentosa*), which is commonly present in fertile soils. The fungus survives over winter in the soil or on grass plants, principally as dormant, dark-colored, compact aggregations of cells called sclerotia. These sclerotia resume growth during warm spring and summer wet periods. Thread-like filaments grow out radially from them through the soil and upon the grass blades during hot, humid weather. Spread from leaf to leaf occurs by the fungus filaments bridging the drops of "dew" or guttated water and by infected leaves contacting healthy ones. The same organism may incite seed, root and crown rot as well as damping-off of young grass seedlings (15).

Recommendations for brown patch control have changed little in the past 25 years, since the early work of Monteith and his associates (11). Mercury chloride mixtures (e.g. Calocure, Calo Clor, Fungchex, Woodridge Mixture No. 1) are commonly sprayed on turf at the rate of 1 to 2 ounces in 5 to 10 gallons of water per 1000 square feet. Preventive applications are required every week or two during the hot summer months. More recently, thiram (e.g. Tersan "50" or "75", Naugatuck Thiram, Stauffer's Thiram Nuggets) has been commonly used, at the rate of 2 or 3 ounces per 1000 square feet, either alone or in combination with mercury chlorides. Field experiments at the Rhode Island Agricultural Experiment Station have shown the mercury chlorides to be more effective than thiram alone under severe disease conditions, and equal to a mixture of mercury chlorides and thiram.

A 3-time spray application program of mercury chlorides prior to weather conditions favorable for disease development apparently killed or inhibited the germinating fungus bodies (sclerotia) which had survived the winter in the soil or on grass plants, thus eliminating the brown patch fungus before injury occurred. Additional ex-

perimentation and golf course superintendents' trials will prove whether a 3-spray, early-season program is sufficient.

If brown patch occurs, a spray containing mercury chlorides alone or in combination with thiram is suggested before the next mowing to help prevent spread of the fungus in infected clippings.

Chemical control of brown patch should be supplemented with good management practices such as removal of dew in the early morning, watering early in the day to permit surface drying before evening, thinning or removing nearby shrubs around pocketed greens to permit better air drainage and to keep greens free of shade, frequent but light fertilizer applications during summer months to avoid a dense vigorous growth, and soil drainage of wet greens (11, 18).

INTRODUCTION

What The Disease Looks Like

Rhode Island Agricultural Experiment Station Bulletin 308 lists over 100 turf diseases, including brown patch, caused by fungi(8). Brown patch, however, probably has been the most publicized because it was the first turf disease on which appreciable research was done and its characteristic damage appears suddenly and strikingly in midsummer during heavy play.

Brown patch may be recognized by a roughly circular brown discoloration of the turf varying from a few inches to several feet in diameter. The disease spreads by growth of a fungus through the soil arising principally from sclerotia located at a central point in the diseased area (8, 13). Under hot, damp conditions the fungus hyphae grow up and over the grass blades forming a fine cobweb-like growth binding the grass blades together (4, 13). Under these conditions a dark "smoke ring" of recently attacked and wilted grass is found as a 0.5- to 1.5-inch border around the diseased area. The smoke ring may advance for one to several days, but later disappears when the weather becomes drier or cooler. The wilted grass turns light brown as the blades dry out (8, 11). These blades have been found to contain large masses of the brown patch fungus mycelium. The cover illustration shows typical active brown patch on Astoria Colonial bent.

Generally only the leaves are attacked, as the conditions favorable for foliage infection do not persist long enough for the fungus

to kill the crown and the roots (8). Infected turf usually recovers and may appear as healthy as ever within two or three weeks. Seemingly uninjured grass blades are commonly found within a diseased patch. How soon the turf recovers depends on the ratio of diseased to surviving "healthy" grass leaves in the infected area (11).

Why The Disease Occurs

At least four factors must be present for brown patch to attack the foliage:

1. The presence of the actively growing fungus in the soil or turf mats.

2. A dense growth of a susceptible grass. The brown patch fungus in greenhouse studies has been shown capable of attacking all grasses tested, including the coarser fescues, and blue, rye and wheat grasses (14). However, the bent grasses, especially Colonial bent, are most commonly attacked.

3. That essentially 100 percent humidity with dew or a film of moisture be present on the foliage.

4. A sustained temperature for several hours of 70 to 90°F. If one of these factors is lacking, brown patch is not likely to be severe. Although all the conditions required to permit the fungus to attack grass crowns and roots are not yet known, infection of underground plant parts can occur at considerably lower temperatures than those required for foliage infection.

Rhizoctonia solani Kuehn, the casual agent of brown patch, is comprised of an indefinite number of pathogenic races. These races vary greatly in their rate of growth, ability to injure grasses, temperature requirements, production of sclerotia, entrance into plants and numerous other factors (10, 11). Even the diseased areas in a single golf green may contain a number of brown patch races which vary greatly in these respects.

How The Disease Spreads

The brown patch fungus was studied to learn how it grows through the soil and over grass blades. Initial infection of the aboveground plant parts was observed to take place commonly through a lower leaf in contact with the soil. The fungus then grew over and

through the grass leaf to others, eventually reaching the uppermost clipped ends. During a brown patch attack the fungus multiplied in the drops of dew and spread rapidly to adjacent plants by bridging in spider web fashion between the drops of water on the grass blade tips (13). During a particular 4-day period favorable to brown patch development, the fungus spread over the mowed surface with the patches increasing about one inch in diameter per day. This daily growth increase was not checked when sprayed with a phenyl mercury fungicide (PMAS) at the rate recommended for crabgrass control.

Greenhouse experiments showed that the fungus grew through soil at a rate of up to one-third inch per day depending on the temperature, fungus race, and density of the grass stand. Other observations indicated that the moisture content, aeration, and nutrient level of the soil may also play a role. Growth through the soil was more rapid at a temperature of 80°F than at 66°F, and at a high seeding rate (3.6 lb. Astoria Colonial bent per 1000 sq. ft.) than a low one (0.25 lb. per 1000 sq. ft.). Evidence indicates that the brown patch fungus obtains nourishment from underground plant parts as it grows through the soil, and that this accelerates its growth.

The causal fungus was also shown capable of being distributed by standard golf green mowers set at ¼-inch cutting height and equipped with a grass catcher. Sufficient clippings escaped the catcher so that inoculum from a single active brown patch, six inches or more in diameter, would be scattered generally over a golf green after several mowings. The fungus was found to remain alive in infected clippings up to four months. Present information indicates that spraying a mixture of mercury chlorides, either alone or in combination with thiram, on active patches before they are mowed will check the disease. Additional research is under way to find more effective and less potentially injurious fungicides for this purpose.

What Perpetuates The Disease From Year To Year

Sclerotia are formed during brown patch attacks and serve as another agent to spread disease on a golf green. They can be spread by players or management practices (e.g. on shoes or equipment). The formation of these fungus resting bodies is important insofar as future brown patch attacks are concerned. Sclerotia are small (generally 1/16 to 1/8 inch long), first a light tan color and later becom-

ing dark brown or black, and formed mostly on grass plants or in the top one-half inch of soil. They are much more resistant than the fragile fungus filaments to drying temperature changes and chemical treatments. Sclerotia of the brown patch fungus have been demonstrated to over-winter and to withstand hot, dry periods. When a sclerotium germinates, fungus hyphae or filaments grow out from its surface through the soil forming an ever-enlarging, roughly circular colony. Figure 1 illustrates how the mold grows out radially from each sclerotium. Under warm, humid conditions the fungus hyphae grow up and over the grass blades webbing them together and spreading from leaf to leaf through the guttated water poised on the tips of the grass blades.

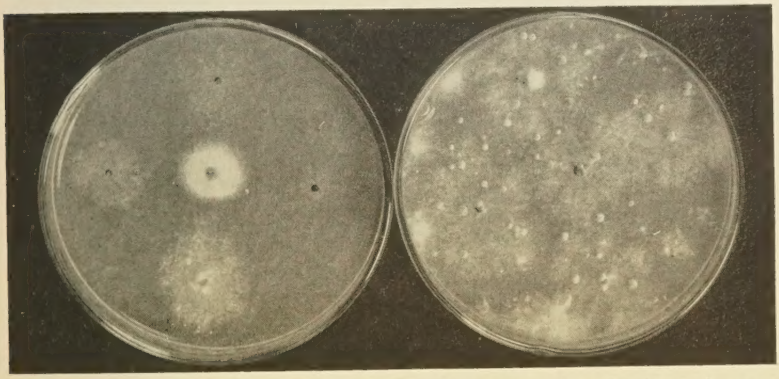

Figure 1—Left; the brown patch mycelium starts from the sclerotia and grows outward in all directions. The dish on the right shows a later stage in the development of the fungus—the formation of new sclerotia.

In laboratory tests, sclerotia were found to be alive after 25 successive germinations over a 9-month period, and to grow vigorously after being dormant for 13 months. This means that a single sclerotium in turf or soil could give rise to a number of separate brown patch infection centers over one or more growing seasons. When temperature and moisture conditions are favorable, the sclerotium resumes activity producing new fungus mycelium which grows out radially through the soil.

Sclerotia are prevalent in most soils, especially slightly acid ones, and may live for four or five years in turf or soil (8). During prolonged brown patch attacks sclerotia may be formed on the grass blades attached to or within the outermost leaf sheath, on the crown,

or in the area where the roots are attached to the crown. Figure 2 shows sclerotia produced on leaves and crowns of Seaside bent following foliage infection. Sclerotia produced near the ground line would escape the mower and remain to initiate future infection centers.

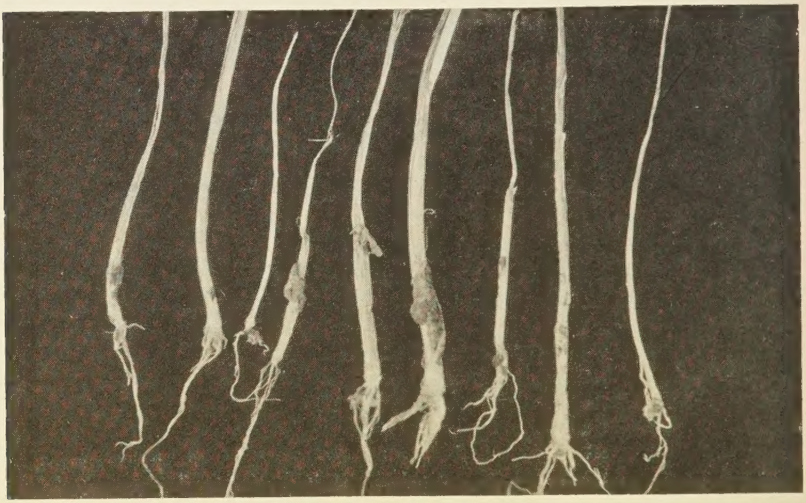

Figure 2—Sclerotia of race 1 of Rhizoctonia solani on crown, crown base and leaf sheath of Seaside creeping bent.

Checking the formation of sclerotia and their subsequent germination should be an important part of a brown patch control program. Without sclerotia to maintain it over winter, the brown patch fungus would likely be killed by cold temperatures or by antagonistic soil organisms able to feed upon it. *Rhizoctonia solani* is considered to be a poor "cellulose competitor" in soil where it has not produced sclerotia, and numerous common soil bacteria and fungi are parasitic or antagonistic to it.

It is necessary to know the conditions required for sclerotium formation and germination. Such factors as host nutrition, soil acidity, temperature, moisture, light, soil aeration, type of soil and racial differences in the fungus itself are important in influencing the number and size of sclerotia produced (1, 16, 17). Sclerotia are formed most abundantly under neutral or slightly acid conditions where the nitrogen supply is high and the carbon low, where the humidity is essentially 100 percent, temperatures are 60-90°F, and where there is an available supply of oxygen. In turf culture, high nitrogen

fertilization frequently results in more severe brown patch (8, 12, 18). This could be the combined result of the nitrogen stimulating a denser, more lush growth of grass which is more prone to infection, and formation of sclerotia which would perpetuate the disease. Recommendations for lessening brown patch should include application of small amounts of nitrogen fertilizer frequently during the season rather than a few heavy applications.

Sclerotia germinate during late spring and summer wet periods when the humidity is at least 98 percent and the temperature at the soil level is 60 F or more for a few hours. Records at the Rhode Island Agricultural Experiment Station turf plots show that even during periods of drought (3 to 4 weeks without rainfall) the turf almost nightly is covered with dew and the air at turf height has a relative humidity of essentially 100 percent. These facts make possible a brown patch control program where chemicals are applied to turf to inhibit sclerotia as they start to germinate and *before* they cause disease, and to prevent a crop of "secondary" sclerotia from forming which may later result in new infection centers. Such a program is aimed at eliminating the primary inoculum at its source —the sclerotium.

GOOD GREENKEEPING TO MINIMIZE DAMAGE

For a great many years golf course superintendents and lawnsmen have carried out a number of practices which help prevent serious outbreaks of brown patch (11, 18). These practices include removing dew in the early morning by brushing over greens with bamboo poles, or the dragging of a mat, hose, rope or similar object over the grass; watering early in the day so that it will be dry before evening; cutting out or pruning of trees and shrubs surrounding pocketed turf areas to supply adequate air drainage; and improving soil drainage to remove surplus water. These precautionary practices remove the dew in which the fungus grows and spreads over the turf (13). Any brown patch control program with chemicals should be supplemented by these cultural practices. The principle of removal of dew to control brown patch was demonstrated a few years ago by Dr. Harry Keil on the Rhode Island Agricultural Experiment Station turf plots (9). Application of surface active chemicals, or wetting agents, prevented accumulation of water in drops on the grass blades for a number of days, thereby effectively controlling brown patch.

Other cultural practices which condition to some extent the amount of disease that occurs are the height of cut, density of stand,

and nutrition. The height of cut on golf greens, generally one-fourth inch, is determined largely by players' preference for a carpet-like putting surface. This requires a dense turf where plants are necessarily crowded. A thick, closely clipped turf is very subject to brown patch (13). Dew forms almost a continuous film of moisture over the turf in which the fungus grows. Dense turf retains more moisture and has a higher humidity at soil level, which is favorable for both sclerotium formation and growth of the mycelium. The effect of host nutrition on sclerotium formation has already been discussed. High fertilization rates (especially of nitrogen) would result in a denser turf with more succulent growth, which is most susceptible to brown patch.

HOW TO ATTAIN CONTROL

The accepted chemical brown patch control measure for the past 25 years has been the application of a mixture of mercury chlorides every 1 to 2 weeks during the warm summer months (8). The mercury chloride mixtures commonly contain two parts of calomel or mercurous chloride and one part of corrosive sublimate or mercuric chloride. This combination has long been favored because it combines the quicker action and higher solubility of corrosive sublimate with the longer residual action of the calomel (11). Although generally giving good disease control, these materials may cause "burning", or chemical injury, when improperly used. To avoid possible injury when using mercury chlorides in hot weather (above 80 F), reduce the dosage by one-half, which is usually sufficient for good protection, and apply the chemical on dull days or in the early evening in at least 10 gallons of water per 1000 square feet and do not wash in. Frequent treatment is required during critical periods when brown patch is active to keep the foliage protected.

During the past 10 years thiram in varying concentrations has been favored by some golf course superintendents. Turf field trials at the Rhode Island and other Agricultural Experiment Stations indicate that when favorable environmental conditions prevail for disease development, thiram often provides poorer control of brown patch than mercury chlorides either alone or combined with thiram (see Table 2).

Based on laboratory and greenhouse studies, several new approaches were taken to the problem of brown patch control. The first was aimed at eliminating the sclerotia, the start for the disease;

the second at internal medication to increase plant resistance by absorption of chemicals which act internally.

How Sclerotia Can Be Killed

A number of chemicals have been used by various investigators in attempts to kill sclerotia. Many of these chemical treatments are impractical for use on turf because of injury to the grass. Several experiments were conducted to test the effect of 10 common turf fungicides and seed protectants for killing or inhibiting formation (or germination) of sclerotia. The chemicals tested in the common dilution used (10 gallons of water for 1000 square feet of turf) were: Acti-dione 1 gm.; Cadminate 0.5 oz.; Calo Clor 2 oz.; Calocure 2 oz.; Captan 50W 2 oz.; Crag "531" 3 oz.; Puratized Agricultural Spray 0.1 pint; Spergon W 3 oz.; Tersan "75" 3 oz.; Vancide "51" 1 pint.

The sclerotia, depending somewhat on their size, shape and degree of dryness, absorbed relatively large quantities of the treatment solutions; up to 25 percent of their original weight.

Three methods were used for exposing the sclerotia to the test chemical solutions: (1) the sclerotia were submerged for 0.5 to 24 hours; (2) sclerotia were sprayed under simulated golf green conditions; or (3) the chemical was dripped on them for 15 seconds. Only the three mercury materials (e.g. Calo Clor, Calocure and Puratized Agricultural Spray) were effective, especially on the smaller and flatter sclerotia. These mercurials were able to act at a distance in the vapor phase, as well as by direct contact. The mercury chlorides were superior in checking the further growth of surviving sclerotia. The poisoning action of phenyl mercury acetate (Puratized) did not appear to be residual. Thiram (Tersan) was valueless in preventing sclerotium germination even when used at the rate of 12 ounces in 10 gallons of water per 1000 square feet—four times the amount suggested for brown patch control—but it did suppress resultant mycelial growth to some extent. Cadmium succinate (Cadminate), like thiram, was ineffective for preventing germination, but suppressed subsequent mycelial growth.

New or "secondary" sclerotia were produced by the mycelia arising from sclerotia which survived treatment, especially from those treated with thiram. This is important as far as future brown patch control in the field is concerned for a single sclerotium which is not killed may give rise to many "secondary" sclerotia, each of which upon germination could result in a new infection center.

Experiments have shown that a large percentage of the brown patch sclerotia found on grasses infected in the field or greenhouse (less than one-tenth inch long with a more or less flat shape) would be killed following a contact period between chemical and sclerotium of only fifteen seconds, if the mercury fungicides were applied at dosages two or four times those commonly used. Such a concentrated mercury material, to avoid turf injury, would have to be applied either in late fall or early spring when temperatures are lower and grasses are dormant and more tolerant to chemical injury, or to the sclerotia as they germinate during late spring or early summer wet periods—a more vulnerable state than when dormant. Recent evidence indicates that chemically injured or killed sclerotia may be readily attacked by competing soil organisms (5). This opens up new possibilities for brown patch control by combining biological and chemical measures. The effects of common soil bacteria and fungi on active sclerotia of *Rhizoctonia solani* have not been fully determined.

Curing Brown Patch; Chemotherapy And Mycelial Eradication

Turf grasses produce new growth from the leaf base, more or less continuously, throughout a growing season. It is almost impossible to keep these new foliage surfaces covered with a protective chemical at all times. If plants could absorb and store some chemical, which would permit them to resist infection for extended periods, great advancement in control would be attained. This type of induced resistance has been called chemotherapy. A chemical which acts internally to check an invading pathogen, reduce symptoms of disease or rid a host of an established pathogen, is termed a chemotherapeutant (7).

In order to prevent disease a chemical is presumed to have entered the plant and either be translocated in an active state to the leaves where infection would normally take place or alter the life processes of the plant.

A simple method was developed for evaluating compounds for brown patch chemotherapy. Four test grasses (Astoria Colonial bent, Seaside creeping bent, Kentucky bluegrass and Creeping red fescue) were planted in soil in quadrants of the same 4-inch pot. Twelve to 14 days later, when the plants were about one-half inch tall, four pots for each treatment were watered with 50 ml. of the test chemical solution for 10 consecutive days. Four reference pots of the grasses

were watered with the same amount of tap water. The liquid was poured into a 2-inch-deep pencil hole in the center of each pot which aided in even distribution of the solution throughout the soil. Care was taken to avoid contact of the chemical solution with the grass foliage. During the 10-day period the grass blades grew approximately 1.5 inches. The day after the last treatment was applied, the foliage was sprayed with a macerated suspension of filaments of the brown patch fungus. The inoculated pots were kept for 48 hours in an incubator which approximated field conditions when brown patch is active (75-88°F and humidity 100 percent).

The combined results of a dozen trials listing the various chemicals and the most effective dilutions used are presented in Table 1 and Figure 3.

The best results were obtained from Tersan "75", Phygon XL, 8-Hydroxyquinoline sulphate (8-HQS), and the antibiotic Endomycin, as Figure 3 illustrates. Other chemicals which indicated promise were cupric maleic hydrazide (CuMH), and 8-Hydroxyquinoline benzoate (8-HQB). Captan 50W gave excellent control, but was injurious within its effective range. Rimocidin sulphate, an antibiotic, caused no visible injury symptoms to the grass plants, but appeared to be more effective in reducing disease at 10 ppm than at 90 ppm. It apparently affected the host's ability to resist the fungus. The remaining chemicals were less effective or valueless as chemotherapeutants, according to the technique employed.

The same chemicals added to a nutrient solution in test tubes were evaluated for their toxicity to the brown patch fungus. Most fungitoxic were the 8-Hydroxyquinoline (8-HQS, 8-HQB) and phenyl mercury compounds; all three of which prevented growth of the fungus at concentrations as low as 4 parts in 10 million. Some of the best chemotherapeutic materials were far less effective. The brown patch fungus grew in solutions containing 400 parts per million of CuMH, 200 ppm of Tersan "75" or Phygon XL, 80 ppm of Calo Clor or Calocure, 16 ppm of Captan 50W, and 10 ppm of Krominate.

In general, no correlation could be detected between the ability of a given chemical to render plants resistant to brown patch damage and its toxicity against the fungus itself. In other words, the chemotherapeutic properties of the compounds appeared to be largely independent of fungitoxicity. This has previously been shown by other investigators (2, 3, 6).

Table 1—Effect of various chemical solutions applied for 10 consecutive days to the soil in pots containing Astoria Colonial bent, Seaside creeping bent, Kentucky bluegrass, and creeping red fescue inoculated with the brown patch fungus.

Chemical	Dilution Used*	Injury**
	ppm	
Neomycin Sulphate	10, 30, **90**	
Netropsin Sulphate	10, 30, **90**	
Acti-dione	**54**, 108	S; tip B and ST at both conc.
Alternaric Acid	10, 30, **90**	
Endomycin	10, 30, **90**	
Rimocidin Sulphate	**10**, 30, 90	
Thiolutin	5, 15, **45**	
Streptomycin Sulphate	90	
Dihydrostreptomycin Sulphate	90	
2-Pyridinethiol, 1-Oxide Sodium Salt	90	
2-Pyridinethiol, 1-Oxide	90	
Dithane D-14	**83**. 167, 222, 667, 2000	M-S; Y & ST
Dithane Z-78	1000	S; Y & ST
Calo Clor	**1000**, 3000	SL-M; Y & ST
Calocure	111, 333, **1000**	
Phygon XL	111, **333**, 1000	ST & Y at 1000 ppm
Tersan "75"	**500**, 1000	SL; ST & Y at 1000 ppm
Thioglycolic Acid Derivative "A"	**111**, 333,1000	SL-M; ST & Y
Thioglycolic Acid Derivative "B"	111, **333**, 1000	Y at 1000 ppm
Thioglycolic Acid Derivative "C"	50, 100, 200, **400**, 1000	
Salicylic Acid	50, **1000**, 3000	S; Y & ST at 3000 ppm
Ferric Ammonium Citrate	111, 333, **1000**, 2000	
Ferrous Sulphate	111, 333, **1000**, 2000	
Cupric Maleic Hydrazide	1000, **2000**	ST at 2000 ppm
Manganous Maleic Hydrazide	1000	SL; ST
N-1-Naphthylphthalmic Acid	333, **1000**	
New Improved Weedone	**333**, 1000	S; ST & B at 1000 ppm
Weedar MCP	333	M; ST
Weedone LV 4	333	S; ST
Hexachloropentadiene, P-162	**62**, 250, 1000	S; B & ST to 100% killing
Captan 50 W	111, 200, **333**, 1000	Tip B all but 111 ppm
Krominate "A"	100	SL; Y & ST
Krominate "B"	111, **333**, 1000	M-S; Y & ST
Krominate "C"	100	
Puratized Agricultural Spray	37, 111, 333, 1000	SL-S; ST & Y
8-Hydroxyquinoline Benzoate	200, **400**, 1000	SL-M; Y & ST at 400 & 1000 ppm
8-Hydroxyquinoline Sulphate	**222**, 400, 667, 1000	SL-S; Y & ST all but 222 ppm
Check, Tap Water		

*Most effective dilution in bold face.
**Injury notes: SL slight; M moderate; S severe; Y yellowing; ST stunting and B=browning.

A second greenhouse experiment was used to evaluate the residual effect of six potential chemotherapeutants. Astoria Colonial plugs two inches in diameter cut from 8-year-old sod were transferred to pots of soil. One month later the grass was trimmed uniformly to a 1-inch height. Then 30 plugs for each treatment were

removed from the pots and soaked in pans containing water solutions of Tersan "75" (500 ppm); 8-HQS (250 ppm); 8-HQB (250 ppm); Phygon XL, first dissolved in a small quantity of ethanol, (333 ppm); Calocure (1000 ppm); CuMH (2000 ppm); or tap water. The plugs were soaked until the soil appeared saturated and then wrapped in aluminum foil to prevent loss of the solution and replaced in the pots. The grass foliage was then lightly sprayed with

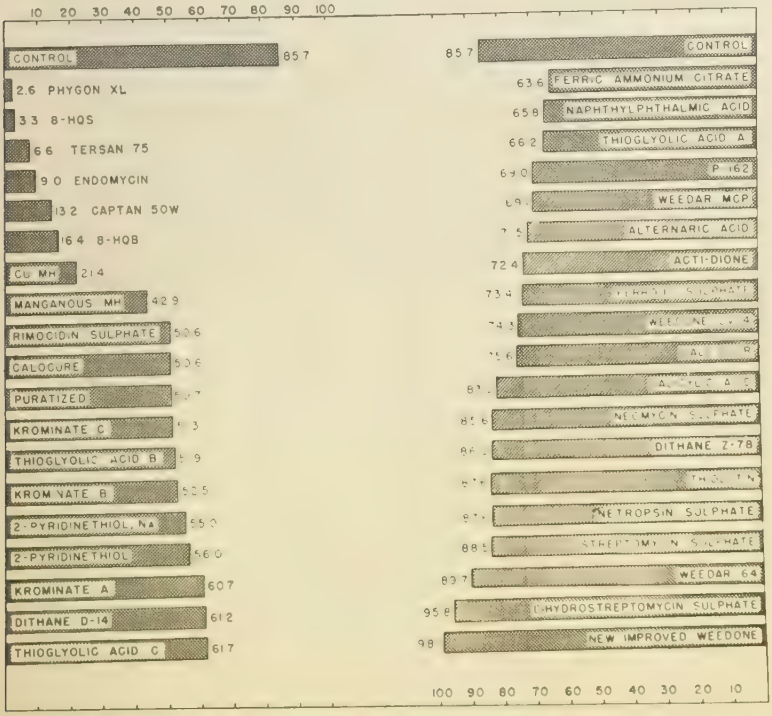

Figure 3—Effect of various chemical solutions applied for 10 consecutive days to the soil in pots containing Astoria Colonial bent, Seaside creeping bent, Kentucky bluegrass, and creeping red fescue inoculated with the brown patch fungus.

the same chemical solution. Five-pot groups treated with each solution were inoculated with the brown patch fungus at 0, 2.5, 5, 10, 20 and 40 days after treatment by pouring on the center of each plug a macerated suspension of the fungus. The inoculated pots were incubated for 48 hours in a humid chamber.

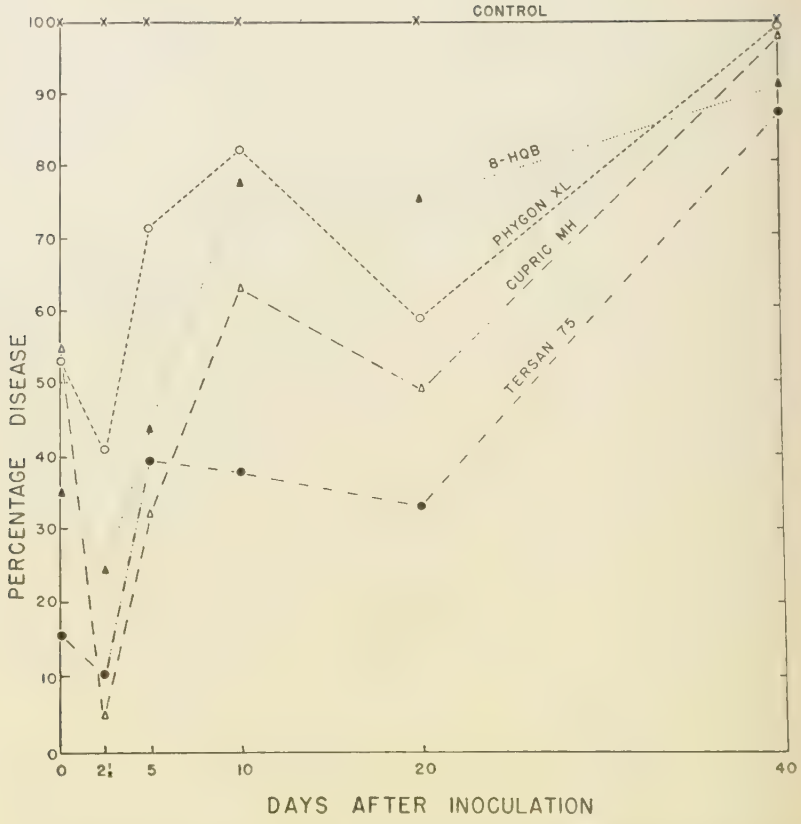

Figure 4—Percentage disease in Astoria Colonial bent plugs inoculated with race 1 of Rhizoctonia solani 0, 2.5, 5, 10, 20 and 40 days after soaking with various chemical solutions.

A summary of the percentage of disease in the plugs, when inoculated 0, 2.5, 5, 10, 20 and 40 days after treatment with the six chemical solutions and the tap water check, is recorded in Figure 4. The severity of disease in the check pots of grass was used as a reference of 100 percent injury. The tap water controls varied from a low of 65 to a high of 92.4 percent infected. The percentages for the treatment solutions were all multiplied by the factor necessary to raise the corresponding check to 100 percent diseased. Consequently, the curves in Figure 4 are plotted from adjusted percentages, with all chemicals maintained in their same relative positions. This

method of presentation gives a truer measure of the residual effectiveness of a given treatment.

The protective action of all chemicals decreased with time. This may be due to dilution of the chemical in the plant, slow breakdown, or possibly an accumulation of the compound in the roots or crowns at the expense of the leaves. All chemicals were more effective in preventing infection 2.5 days after treatment than at any other time. This is believed due to the chemicals having been absorbed and translocated within 2.5 days after treatment.

The best results were obtained during the period from 2.5 to 20 days after treatment using Tersan "75" and cupric maleic hydrazide. Tersan provided somewhat better disease protection. The dose of inoculum administered to each plug was considerably greater than would conceivably occur normally in the field. This experiment suggests that Tersan "75" and cupric maleic hydrazide could protect turf, for 20 days at least, when used as a soil drench before brown patch is evident. Calocure and 8-HQS (Hydroxyquinoline sulphate) were omitted from Figure 4 to simplify the chart, since they were similar in action to 8-HQB and Phygon XL.

What Type Of Treatment Controlled Brown Patch Best In 1953?

Prior tests indicated that none of the materials being used to control brown patch completely checked the disease. A number of fungicides—some new and some old—were tested during the 1953 growing season against brown patch at the Rhode Island Agricultural Experiment Station turf plots. Two areas were used: (a) Astoria Colonial bent severely injured by the disease in 1952; and (b) Rhode Island Colonial bent relatively free of brown patch in 1952. The Rhode Island bent area was "seeded" with sclerotia, to insure uniform inoculation. After mechanically spiking, three sclerotia per square foot were punched one-fourth inch deep into the "spiker" holes. The turf was watered weekly with an oscillating-type overhead sprinkler to favor severe brown patch attack.

The treatments were of three types:

(a) Regular sprays (R) applied at 2-week intervals starting June 12 on Astoria and June 26 on the Rhode Island bent.

(b) Three sprays (R-3) of Calocure or Calo Clor, applied a week apart, starting June 26 to the Rhode Island bent.

(c) Chemotherapeutic soil drenches (S) applied on June 26 and July 24 to provide a total of 0.85 inches of water to the turf.

Each fungicidal treatment was applied to five randomized 5 x 5-foot plots. Sprays were applied with a conventional nozzle at about 100 pounds pressure using a 30-gallon power sprayer. Treated areas were mowed an average of three times a week at $^1/_4$-inch height. The June 26 application was made during dry, bright weather with the

Table 2—Percent brown patch control and injury with fungicides on Astoria and Rhode Island Colonial bents at the Rhode Island Agricultural Experiment Station turf plots in 1953.

Fungicide	Type of Spray Applic.[1]	Dosage per 1000 Sq. Ft. in 10 Gal. Water	Brown Patch[2] 7/22	8/17	8/18[3]	Injury[2] 6/28	7/9[3]
		Ounces		Percent			
Astoria Colonial Bent (natural inoculum)							
Check (no fungicide)			39[4]	25	41	15	1
Calocure	(R)	2	1	0	3	3	0
Calocure & Tersan "75"	(R)	1 each	1	1	2	6	0
Tersan "75"	(R)	2	0	5	15	6	0
Captan 50W	(R)	2	3	8	17	5	1
Tersan "75"	(S)	35.7	1	9	20	15	0
Phygon XL & Nugreen	(S)	23.8 & 53.3	28	22	31	1	0
Rhode Island Colonial Bent (inoculated with sclerotia)							
Check (no fungicide)			7		11	0	3
Calocure	(R-3)	2	0		3	0	0
Calo Clor	(R-3)	2	0		2	3	9
Calo Clor	(R)	1	0		15	4	7
A-935	(R)	0.2	3		15	1	4
MCW-84	(R)	1	2		19	0	3
Crag 531	(R)	3	0		25	2	3
Liquiphene	(R)	0.33	3		21	3	3
Krominate	(R)	0.5	8		14	0	2
Endomycin	(R)	0.023	7		20	0	1
Acti-dione	(R)	0.5	13		24	2	2
Tersan "75"	(S)	35.7	2		1	1	0
Phygon XL	(S)	23.8	3		6	0	2
Cadminate	(S)	11.9	0		9	1	5
Krominate	(S)	5.49	5		9	2	3
8-HQB	(S)	17.84	3		16	0	2
Nugreen	(S)	53.3	22		1	0	0
PS-242	(S)	12.0	9		26	0	4
Calo Clor	(S)[5]	35.7 then 11.9	0		2	41	46
Captan 50W	(S)	11.9	8		10	1	2
CuMH	(S)[5]	71.4	0		100	8	100

[1](R) = regular spray schedule; (R-3) = 3 sprays applied for season, June 26, July 3, and July 10; (S)=soil drenches applied June 26 and July 24.

[2]Average of 5 randomized plots, 7 applications at 14-day intervals.

Notes for 7/9 and 8/18 include brown patch, and "off-color" due to "fading-out" caused by **Curvularia** sp. and other diseases.

[4]Percentage area covered by brown patch or injured by chemicals.

The concentration of the Calo Clor (S) was reduced for the July 24 application because of severe injury. The second soil drench of CuMH was not applied because the turf was 100 percent dead.

average temperature 79 F during the treatment period and a maximum of 89 F the following day. This was partially responsible for the injury reported in the tabulated data (Table 2). Other turf troubles were active, however, for the Astoria bent control plots sprayed only with water showed 14.6 percent "injury" or "off-color" on June 28. The corresponding Rhode Island bent control plots showed no injury at this time.

Brown patch was evident and severe on the plots only twice during 1953; July 22-24 and August 17-21. Both attacks occurred just before treatment was scheduled. A closer spacing of treatments might have prevented the disease from occurring on some plots. The turf recovered between the two attacks and appeared healthy about two weeks after the active phase of the disease occurred.

Noteworthy results evident in Table 2 are almost complete disease control on Astoria bent using Calocure (2 oz. in 10 gal. per 1000 sq. ft.) or a Calocure-Tersan "75" mixture (1 oz. each in 10 gal. per 1000 sq. ft.). The Calocure in the mixture seemingly is the more active material, for Tersan "75" alone (2 oz. in 10 gal. per 1000 sq. ft.) gave relatively poor control when notes were taken on August 17-18. The rapidity with which the fungus can cause damage was seen on the untreated Astoria bent plots where the percentage of area diseased increased from 25.4 to 41 percent within 24 hours (August 17-18). Correspondingly, disease in the Tersan-, Captan- and Phygon-treated plots increased 10 percent, while the more potent treatments—Calocure and the Calocure-Tersan mixture—permitted only an increase from 0.4 to 3 percent and from 0.6 to 2.0 percent respectively.

Several fungicides gave almost perfect control on the Rhode Island bent where the disease was less severe. The "3-time" spray program (R-3 of Calocure or Calo Clor resulted in excellent disease control. These sprays were aimed at killing the brown patch sclerotia as they germinate during rains before soil temperatures reach 70 F, and at preventing spread of the fungus mycelium through the soil. The "3-time" spray program evidently killed the fungus in the soil for plots were essentially free of brown patch 40 days after application of the last spray. Three "early season" applications of Calocure or Calo Clor gave better control of brown patch than did 7 or 8 applications of these same chemicals applied at 2-week intervals during the brown patch season.

Three successive weekly applications of fungicide, each following watering by 48 hours, were probably successful in controlling brown patch for the following reasons:

a. The sclerotia, which are near the soil surface, absorbed water, germinated, and the halo of mycelial growth was destroyed by the first application of mercury chlorides. After the next sprinkling the sclerotia again germinated and the resulting new mycelial growth was again destroyed upon application of the second treatment. The food supply in the sclerotia, which may be considered analogous to that in a plant seed, was reduced and the vitality of the sclerotia weakened.

b. Laboratory experiments have shown that a number of common soil-borne fungi are capable of growing on brown patch sclerotia. These competitive organisms were frequently as effective in killing injured sclerotia as were applications of mercury chlorides. Fungi have been found attacking sclerotia after treatment with mercury chlorides, thiram, and mixtures of these two chemicals.

c. In actual practice the early season alternating schedule of watering and treating with mercury chlorides, coupled with the attack on the weakened sclerotia by predaceous soil micro-organisms, effectively destroys the brown patch sclerotia and thus prevents the disease.

Apparently applications of mercury chlorides at standard 2-week intervals, with watering in between, is not as effective as treatments a week apart. The mycelium from the germinating sclerotia may have penetrated grass tissues and become relatively safe from the lethal action of the chemical, or spread sufficiently through the soil so as to be difficult to reach or eradicate.

Golf course superintendents with a knowledge of when and where the sclerotia germinate can so arrange their watering and spraying schedule to prevent brown patch with fewer fungicide applications. An appreciable saving is possible in the cost of fungicides for the season, the expense of labor involved in applying the sprays, and in avoiding turf injury by making applications early in the cooler season.

Treatment with Acti-dione-ferrated (0.5 oz. in 10 gal. per 1000 sq. ft.) and Nugreen as a soil drench (53.3 oz. per 1000 sq. ft.) resulted in considerably more disease on the Rhode Island bent than the untreated controls during the first brown patch attack on July

22. Treatment with a combination of Nugreen (46 percent nitrogen urea) and Phygon XL increased greatly the susceptibility of turf to brown patch. As previously noted, heavy nitrogen fertilization should be avoided during hot weather when brown patch may be prevalent.

When brown patch is active, and the fungus is invading the grass blades, the fungicide should not be watered in after application. As a preventive, before the disease develops and when the sclerotia start to germinate, watering following spray or dry application will carry the fungicide from the leaf blades to the top half-inch of soil where the majority of the sclerotia are located. Watering in of the fungicide depends upon where the fungus is and its developmental stage.

The third type of treatment (i.e. soil drenches) was applied twice during the season. Ten different compounds were compared. The results were generally disappointing. Such high dosages were required that this method appears impractical at present. Soil drenching required application of chemicals at rates varying from 12 to 71 ounces, while the standard spray (R) program required from 0.16 to a maximum of 14 ounces for the brown patch season. More field trials are required to work out better means of applying the chemicals, safe dosages to use, effective length of the protective period, and other factors. Plots drenched with Tersan "75" (35.7 oz. per 1000 sq. ft.) averaged 9.9 percent of their area damaged by brown patch, while plots sprayed eight times at 2-week intervals with the same compound (2 oz. in 10 gal. per 1000 sq. ft.) averaged 6.9 percent brown patch compared with the untreated controls at 35.1 percent. It is unfortunate that active brown patch did not occur sooner after treatment to validate the greenhouse data.

The treatments giving noticeable injury were Calo Clor (S), Calo Clor (R), Calo Clor (R-3), CuMH (S), Liquiphene (R), Krominate (S) and Acti-dione-ferrated (R). Calo Clor and Liquiphene are both mercury-containing chemicals which are known to be injurious when improperly applied in hot weather. No noticeable injury occurred using Calocure. Injury was difficult to evaluate later than June 28 because of the presence of other diseases, particularly "fading out" incited by *Curvularia* sp. The untreated control plots were "off-color" to some extent in contrast to the plots sprayed with Nugreen, Calocure and Tersan "75" which stimulated grass growth

for about a 2-week period. Turf treated with these compounds appeared healthier and darker-green than the others.

What These Studies Indicate

Mercury chlorides, with or without the addition of thiram, will continue to be the primary control for brown patch. A schedule directed at killing the sclerotia while they are actively serving as a food source for advancing filaments should be tried by golf course superintendents. Watering turf prior to applying a mercury chloride fungicide, aimed at killing or weakening the sclerotia, appears to be a meritorious practice. A saving in labor and materials is possible, while fungicide applications prior to hot weather will minimize likelihood of turf injury. Another possibility is the use of chemicals which grasses will absorb to acquire increased resistance to disease. Selection of bent strains more resistant to brown patch should naturally supplement chemical or cultural control measures.

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